REMARKS

Reconsideration is requested.

Claims 1-51 are pending. Claims 4, 7, 8, 12 and 17-48 have been withdrawn from consideration.

The Examiner's indication that claims 11 and 13-15 contain allowable subject matter is acknowledged, with appreciation. See page 4 of the Office Action dated May 13, 2008.

The Section 103 rejection of claims 1-3, 5-6, 9-10, 16 and 49-51 over Baird (U.S. Patent No. 6,037,329), Hanai (U.S. Patent No. 5,952,472) and Owen (Journal of Immunological Methods, 1994, 168:149-165) is traversed. Reconsideration and withdrawal of the rejection are requested in view of the following distinguishing comments.

The Examiner states as follows with regard to the primary reference:

> "It is undisputed that Baird et al. teach using an inhibitor of FGF-8 for therapeutic purpose (see, e.g. column 25 first full paragraph). Baird et al. also teach that members of the FGF family mediated pathological conditions such as arthritis (see, column 10, last paragraph)." See page 3 of the Office Action dated May 13, 2008.

The applicants respectfully disagree with the Examiner's statements regarding the teachings of the cited primary reference. Specifically, the applicants understand Bard et al. to teach using inhibitors at bFGF for inhibiting smooth muscle cell proliferation and angiogenesis, but not the therapeutic purpose. The applicants believe that Bard et at does not teach using inhibitors of all FGF family molecules for

therapeutic purpose. The applicants note that the first full paragraph of column 25 of the patent, cited by the Examiner, includes the following:

> Particularly useful antisense nucleotides and triplex molecules are molecules that are complementary or bind to the sense strand of DNA or mRNA that encodes a protein involved in cell proliferation, such as an oncogene or growth factor, (e.g., bFGF, int-2, hst-1/K-FGF, FGF-5, hst-2/FGF-6, FGF-8). Other useful antisense oligonucleotides include those that are specific for IL-8 (see, e.g., U.S. Pat. No. 5,241,049; and PCT Applications WO 89/004836; WO 90/06321; WO 89/10962; WO 90/00563; and WO 91/08483). These nucleic acids or nucleic acids that encode antisense can be linked to bFGF for the treatment of psoriasis. Antisense oligonucleotides or nucleic acids encoding antisense specific for nonmuscle myosin heavy chain and/or c-myb (see, e.g, Simons et al. (1992) Circ. Res. 70:835-843; PCT Application WO 93/01286, U.S. application Ser. No. 07/723,454: LeClerc et al. (1991) J. Am. Coll. Cardiol. 17 (2 Suppl. A):105A; Ebbecke et al. (1992) Basic Res. Cardiol. 87:585-591) can be targeted by an FGF, for example to inhibit smooth muscle cell proliferation, such as occurs following angioplasty.

The applicants believe that Baird et al. also teach the function of FGF family and pathological role of FGF family, for example, in tumor development, rheumatoid arthritis, proliferative diabetic retinopathies and other complications of diabetes, as noted in the following column 10, last paragraph of the patent, also cited by the Examiner.

> FGFs exhibit a mitogenic effect on a wide variety of mesenchymal, endocrine and neural cells and are also important in differentiation and development. Of particular interest is their stimulatory effect on collateral vascularization and angiogenesis. In some instances, FGF-induced mitogenic stimulation may be detrimental. For example, cell proliferation and angiogenesis are an integral aspect of tumor growth. Members of the FGF family, including bFGF, are thought to play a pathophysiological role, for example, in tumor development, rheumatoid arthritis, proliferative

> diabetic retinopathies and other complications of diabetes. To reduce or eliminate mitogenesis, muteins of FGF are constructed as described below. Such muteins retain the ability to bind to high and low affinity receptors.

The applicants believe however that Baird et al do not teach that FGF-8, one of the FGF family, is related to rheumatoid arthritis.

The applicants believe that one of ordinary skill in the art would not have recognized from the cited art that any FGF molecule is related to any pathological condition. Therefore, the applicants believe that it would not have been obvious for an ordinarily skilled person in the art to have predicted a role of FGF-8 in the pathological condition of rheumatoid arthritis.

The applicants submitted that the secondary art fails to cure these deficiencies of the primary reference and that the claims would not have been obvious over the combination of the cited Bard et al., Hanai et al. and Owen.

The claims are submitted to be in condition for allowance and a Notice to that effect is requested. The Examiner is requested to contact the undersigned, preferably by telephone, in the event anything further is required in this regard.

Respectfully submitted,

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